REMARKS

Claims 22-25 are pending in this application. All of the claims stand rejected. Claim 22 has been amended to recite mutations which result in cardiac arrythmia, a solution suitable for measuring the conductance of a K+ channel current and mutations which include a leucine at amino acid residue 29. It is believed that these amendments do not constitute new matter and their entry is requested.

35 U.S.C. 112 enablement rejections

Claims 22-25 were rejected under 35 U.S.C. § 112, first paragraph for lack of enablement. The Examiner is of the opinion that the specification does not support enablement of methods to screen for drugs that are useful in treating any disease. The Examiner is further of the opinion that the state of the art at the time of filing only linked HERG mutations to LQT syndrome, and not other diseases. The claims have been amended to recite methods for screening for drugs useful in treating cardiac arrythmias. Support for this amendment can be found, *inter alia* at the bottom of page 53 thru page 55, where it is noted that mutations in HERG are shown to induce cardiac arrythmias.

Claim 25, directed to cells expressing HERG from a transgenic animal, remains rejected because the Examiner is of the opinion that it is unclear whether the specific mutations disclosed would exhibit altered K+ channel current in the transgenic animal or cells because the cells "already have the wildtype HERG in its genome." The Examiner further asserts that the disclosed mutations are in human HERG gene, that the human and mouse HERG are not identical and thus that it is unpredictable whether mutation in the same site in the mouse gene would alter K+ channel function. In response, it is respectfully submitted that the Examiner has not met the burden of providing specific technical reasons as to why the claim would not be enabled. Assuming, arguendo, that the human and mouse HERG are not "identical," the Examiner has not provided any evidence as to why the disclosed mutations in conserved functional regions would not function in the mouse in the same manner. Furthermore, a comparison of the human sequences disclosed in the present application with the *mus musculus* potassium voltage gated channel, subfamily H (Accession No. GI:730202) showed a greater than 95% homology between the two protein sequences.

The Examiner has also asserted it is unpredictable whether the disclosed mutations would alter K+ channel current in the mouse or other transgenic animal, since the animal would have a wildtype HERG present in its genome. In response, it is submitted that Examiner as provided no

CONCLUSION

In view of the above amendments and remarks, it is believed that the claims satisfy the requirements of the patent statutes and reconsideration of the instant application and early notice of allowance are requested. The Examiner is invited to telephone the undersigned if it is deemed to expedite allowance of the application.

RESPECTFULLY SUBMITTED,					
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